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THE
LARYNGOSCOPE.

VOL. LIII SEPTEMBER, 1943. No. 9

THERAPY OF DEAFNESS.

PART III — REPORT OF CASES.*

DR. LOUIS GUGGENHEIM, Los Angeles.

In the two preceding divisions of this paper,[1,2] I discussed the hearing apparatus, audiometery and voice testing, prophylaxis and pathology of deafness. In the present division I am presenting the results of treatment in children under 13 years of age.

Since the commonest cause of deafness in children is tubal obstruction from adenoid and unresorbed mesenchyme and since each year brings the child nearer to permanent tympanal fibrosis, the earlier such an impairment is discovered and treated the better is the prognosis.

It seems that the age of 13 is a rough dividing line between reversible early tympanal fibrosis and irreversible fibrous tissue formation. So very often moderate impairment of hearing is not discovered until it is too late for cure. If parents could be persuaded to have hearing tests made on their children, before starting to school, so much more would be accomplished than by leaving it to the schools to discover the defect.

Would it not be an excellent plan for all schools to require, before admission, a certificate of examination from a pediatrician, an ophthalmologist and an otolaryngologist? After admission and at certain intervals the hearing should be retested. It is so common for moderate impairment of hearing

*From Department of Otolaryngology, University of Southern California.
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to be overlooked and the child subjected for years to psychologic damage that we should not depend upon parental observation, but carefully test with voice and audiometer every single child before he faces the difficult adjustments of school life.

Again I want to emphasize that the otolaryngologic examination should always include the direct inspection of the nasopharynx with the Andy Love retractor. Taking for granted that the adenoid has been removed because the tonsillar fossae are clean and the nasal breathing free has resulted in thousands of unnecessary cases of deafness.

Crowe[3] states, "An interesting fact about the nasopharynx was brought out by a study of 1,365 school children during the past year. All of the children were between the ages of 8 and 14 years. In 755 the tonsils and adenoids had been removed at various hospitals in Baltimore. As a rule the tonsils had been cleanly removed, and in many the oropharynx looked normal; but more than 75 per cent had a marked recurrence of adenoid tissue in the nasopharynx. This was due not to incomplete operation but to the fact that in this area lymphoid tissue is an integral part of the mucous membrane. It cannot possibly be removed in its entirety unless the entire thickness of mucous membrane is taken out, a procedure which is manifestly impossible." He then continues, "The proper treatment is irradiation, not a second operation."

Crowe believes that for many reasons Roentgen rays should not be used but that a local application with the radon nasal applicator solves the problem. He adds, "If used carelessly or if entrusted to a technician, radium therapy may be extremely dangerous."

Although adenoid tissue can and does recur, it has been my experience that where a clean tonsillectomy has been done and adenoid tissue is present, it is usually because it has never been properly removed. Since doing the direct operation I have seen very few recurrences of sufficient size to obstruct the nose or Eustachian tubes.

If Crowe's radon nasal applicator proves successful upon being checked by a sufficient number of otolaryngologists, it may displace the surgical procedure described by me.

At present the danger both to the otolaryngologist and the patient from the use of radium, the proximity to the pituitary, the possibility of the eventual development through metaplasia of nasopharyngitis sicca, makes me hesitate to try it. Others, more courageous, will give us their experiences in time and a final and proper evaluation of the method will be possible.

In Table 1 are presented 36 children, all 13 years of age or younger, who have been under treatment for impaired hearing resulting from tubal obstruction. In addition to the treatment appearing in the table there was in each a dietary study and regulation, which included the prescribing of adequate vitamins for oral use.

In Table 1 it will be seen that in 12 cases (33 per cent) there was a family history of deafness. It should be kept in mind that this positive family history could refer to otosclerosis and degenerative labyrinthine deafness, but also it could mean the exuberant adenoid and mesenchyme type.

KEY TO TABLE 1.

Previous Sickness: (A) allergy, (O) otitis media, (M) measles, (C) chickenpox, (P) pertussis, (I) influenza (Mu) mumps, (D) diphtheria, (H) head injury, (Q) quinine to mother to induce labor, (S) scarlet fever (Pn) pneumonia, (F) frequent colds, (H.T.) hypothyroid.

Family History: Impaired hearing, otitis media. (P) positive, (N) negative.

Nose and Throat: (S) sinusitis, (T) chronic tonsillitis, (L) lymphoid tissue obscuring tubal orifices, (Tl) lymphoid rests in tonsillar fossae, (A) allergy.

Drum Membrane: (N) normal, (R) retracted, (Mf) malleus fixed or mobility impaired, (Mn) malleus mobility normal, (S) evidence of serous transudate.

Vestibular: (N) normal reaction to turning, (%) per cent loss, R and L.

Operation: (T + A Tu) tonsillectomy, direct adenoidectomy and tubal dilatation with Gerjoye instrument; (2nd T) tonsillar rests removed; (A Tu) direct adenoidectomy and tubal dilatation.

Case	Age	Sex	Previous Sickness	Previous Operation	Family History	Nose & Throat	Drum Membrane, Right	Drum Membrane, Left	Vestibular	Air Con. Decibel Loss Speech Range, Average R.
1—P.S.	6	M	O, P, M, Mu	T & A	P	L	R, Mf	N	N	10
2—M.C.	10	F	O, H, A, M, C, P	None	N	T, L, Thornwald's cyst	N, Mn	N, Mn	R-50% L-30%	32
3—F.K.	10	M	O	T & A	P	L	N, Mn	N, Mn	R-50% L-50%	34
4—H.S.	6½	M	F	T & A	N	L, Tl	N, Mf	N, Mf		22
5—J.B.	9	F	A, H, T.	T & A twice	N	Tl, L, A	R, Mn	N, Mf	R-30% L-30%	66
6—G.M.	12	F	A, Q, P, Mu, M, C, I	T & A	P	L	N, Mn	N, Mn	R-50% L-50%	45
7—D.B.	7	F	F, M, C, Mu, O, P	None	N	T, L	R, Mf	R, Mf S	N	18
8—Dl. B.	7	M	Q, Mu, P, F	T & A	P	L, Tl	R, Mf	R, Mf	N	16
9—H.G.	7	M	O	T & A	N	L	Thickened, scar, Mf.	R, Mf		10
10—J.H.	10	M	Q, M, C, O, S, Mu, P	T & A	N	S, Tl, L	R, Serous transudate picture	R, Serous transudate picture	R-50% L-50%	9
11—A.H.	10	F	M, C, I, O, Mu, D	T & A	P	Tl, L	R	R, Mf		7
12—B.H.	11	M		None	N	T, L	R	R, Serous transudate picture		25
13—R.H.	6	F	O, M, Mu, P, Pn, I, F	None		T, L	R, Mn	R, Mn	N	15
14—M.I.	11	F	O, A, M, P, Mu	None	N	T, L	R, Mf, Serous transudate picture	R, Mf, Serous transudate picture		15
15—P.J.	6	M	M, C	None	N	T, L	N	mucousexudate picture		30
16—B.J.	7	F	Q, O, S, Mu, P	T & A	N	S, L	Dry perforation	Dry perforation		48
17—G.Mc.	6	M	M, O	Cleft Palate T & A	N	L	R, Mf	Dry perforation. Mf.	R-50% L-50%	85
18—D.M.	5½	M	O, S, Q, F	None	N	T, L	R. Evidence of serous transudate	R, Mf	N	18
19—C.M.	12	F	O	T & A	N	L	R, Scar Mf	R, thickened Mf	R-30% L-30%	43
20—Cl.M.	5	M	Q, O, F	None	P	T, L	R, Mf	R, Mf		40
21—L.M.	12	M	P, M, Mu, I	T & A	P	Negative	R, atrophic, Mf	R, atrophic, Mf		38
22—Ge.M.	5½	M	O, Pn, S	T & A	N	Negative				39
23—C.R.	10	F	F, P	None	N	T, L	N, Mn	N, Mn		6
24—J.R.	8	M	Q, C, P, Mu, M	None	N	T, L	R, Mf	R, Mf	N	17
25—E.S.	9	F	O, M, C, P	None	N	T, L	R, Mn	R, Mn	N	18
26—K.S.	9	F	O, A, C, M, Mu, P	T & A	P	L	N, Mn	N, Mn		23
27—J.S.	13	M	M, C, Mu	T & A	N	L	N, Mn	Mf		9
28—R.S.	8	M	M, Mu, O	T & A	N	Tl, L	N, Mn	N, Mn		11
29—T.S.	12	M	Shocked by Explosion. S, O, I. Father, Wassermann, positive P	T & A	N	Negative	N, Mn	N, Mn	N	66
30—V.V.	5	F	O, M, I, P, A	T & A	P	Tl				24
31—J.V.	9	M	Quinine for colds Chr. nephritis, M	T & A	N	L	N, Mn	N, Mn		
32—F.Y.	10	M	O, Pn, P, C, M, H. T.	T & A	P	Tl, L	R, Mf	R, Mf		13
33—R.B.	5	M	O, A	5 bilateral myringotomies	P	T, L	R, Mf Atrophic	R, Mf Atrophic		17
34—B.T.	6	F	O, M, P	None	N	T, L	R, Mf	R, Mf		71
35—W.T.	8	M	O, A, M, P, F	None	P	T, L	R, Mf	R, Mf	N	33
36—L.T.	6	F		None	N	T, L	N	N	R-60% L-60%	85

Case	Air Con. Decibel Loss Speech Range, Average, L.	Operation	A. T. or A. O. Air C. Decibel Loss Speech Range, Average, R.	A. T. or A. O. Air C. Decibel Loss Speech Range, Average, L.	Decibel Gain Right	Decibel Gain Left	Masking, Whispered Voice at Exam.	Masking, Whispered Voice Final
1—P.S.	9	A, Tu	0	0	19	9		
2—M.C.	89	T + A, Tu, Thornwald's cyst removed	3	65	29	24		
3—F.K.	5	A, Tu	1	0	33	5		
4—H.S.	19	2nd, T, A, Tu	8	11	14	8		
5—J.B.	27	2nd, T, A, Tu	66	15	0	12	right 1 ft. 4 in. left 5 ft. 6 in.	right 2 ft. 9 in. left 11 ft. 4 in.
6—G.M.	43	A, Tu	27	29	18	14	right nil left nil	right 7 ft. 5 in. left 7 ft. 2 in.
7—D.B.	16	T + A, Tu	2	5	16	11		
8—Dl.B.	32	2nd, T; A, Tu, 1-yr. later, A, Tu, 1-yr. later 3rd, A, Tu	0	0	16	32	right 6 in. left 6 in.	right 7 ft. left 27 ft.
9—H.G.	9	A, Tu	3	above normal	7	20		
10—J.H.	16	2nd. T, A, Tu	3	0	6	16		
11—A.H.	18	2nd. T, A, Tu	0	4	7	14		
12—B.H.	23	T + A, Tu	11	12	14	11		
13—R.H.	19	T + A, Tu	11	4	4	15		
14—M.I.	30	T + A, Tu	above normal	9	22	21		
15—P.J.	35	T + A, Tu	6	1	24	34		
16—B.J.	51	A, Tu	18	9	30	42		
17—G.Mc.	73	A, Tu	61	57	24	16		
18—D.M.	10	T + A, Tu	above normal	0	27	10		
19—C.M.	42	A, Tu	0	above normal	43	47		
20—Cl.M.	34	T + A, Tu	8	8	32	26		
21—L.M.	50	Tu, Twice	7	1	31	49	right 5 ft. left 7 ft.	right 27 ft. left 22 ft.
22—Ge.M.	24	Tu	12	7	27	17		
23—C.R.	1	T + A, Tu	above normal	above normal	18	10		
24—J.R.	45	T + A, Tu	1	19	16	26		
25—E.S.	13	Tu, T + A	4	6	14	7		
26—K.S.	17	A, Tu	9	20	14	0		
27—J.S.	13	A, Tu	above normal	above normal	21	22		
28—R.S.	8	2nd. T, A, Tu	1	0	10	8		
29—T.S.	41	None	37	38	29	3		
30—V.V.	20	2nd. T, Tu	above normal	above normal	34	36		
31—J.V.		A, Tu			audiometry unchanged	audiometry unchanged		
32—F.Y.	13	2nd. T, A, Tu	6	above normal	7	16		
33—R.B.	23	T + A, Tu	above normal	3	21	20		
34—B.T.	57	T + A, Tu	25	7	46	50		
35—W.T.	27	T + A, Tu	16	16	17	11		
36—L.T.	92	T + A, Tu	46	50	39	42		

(A. T. or A. O. = A. T.) after treatment with dietary regulation and vitamins. (A. O.) after operation.

ANALYSIS OF TABLE 1.

Of the 36 cases, 21 are males, 15 females. Twenty-four of the 36 (66 per cent) gave a history of one or more attacks of otitis media. In seven cases (19 per cent) quinine had been administered to the mother to induce labor. Twenty-one (58 per cent) had had tonsillectomy and adenoidectomy performed. In 12 cases (33 per cent) the family history of deafness was positive. In 15 (41 per cent) there was chronic tonsillitis. In 32 cases (88 per cent) nasopharyngeal lymphoid tissue was found obstructing the tubes. In only one case was there evidence of nasal allergy at the time of examination, although seven cases gave a history of having had treatment for nasal allergy. There were two cases of sinusitis. In 15 cases tonsillectomy, direct adenoidectomy and tubal dilatation was performed. In 17 cases direct adenoidectomy and tubal dilatation alone was performed. In one case there was recurrence of adenoid which required in all, three adenoidectomies. In two cases tubal dilatation alone was performed. In one of these, the dilatation was repeated.

The 36 children showed an improvement of 1,433 dcb, an average of 39.8 dcb. gain for each. The total gain for right ears was 729 dcb. (average for each, 20.2 dcb.); for left ears, 704 dcb. (average for each, 19.5 dcb.). Two right ears and two left ears showed no audiometric improvement.

Whereas in adults and older children the voice test is invaluable, young children are often very unsatisfactory in their response because of fatigue, timidity, etc.

In the audiograms it will be noted that bone conduction was tried for 8,192 in certain instances. This is now considered valueless as it is impossible with our present bone conduction receiver to exclude this high frequency from being conducted through air.

REFERENCES.

1. GUGGENHEIM, LOUIS: Therapy of Deafness, etc. THE LARYNGOSCOPE, 53:7:441-456, July, 1943.

2. GUGGENHEIM, LOUIS: Therapy of Deafness, etc. THE LARYNGOSCOPE, 53:8:503-518, Aug., 1943.

3. CROWE, S. J.: *Arch. Otolaryngol.*, 33:4:618-622, April, 1941.

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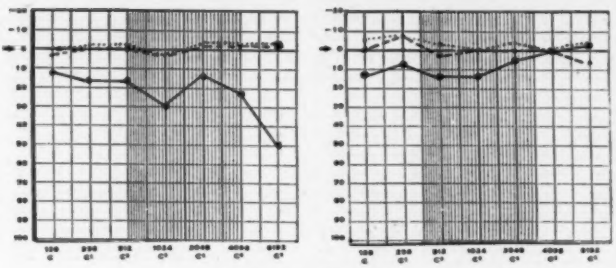
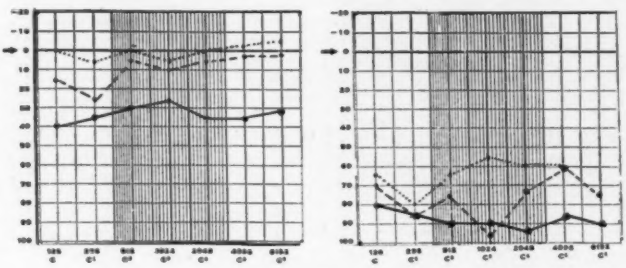


Case 1. P. S. Age 6.

Right
Air Conduction—Feb. 18, 1939
- - - May 27, 1939
. . . . Sept. 2, 1939
Op. A. Tu. March 1, 1939

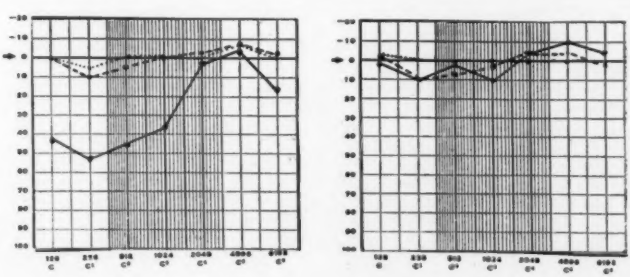
Left
Air Conduction—Feb. 18, 1939
- - - May 27, 1939
. . . . Sept. 2, 1939
Op. A. Tu. March 1, 1939



Case 2. M. C. Age 10.

Right
Air Conduction—April 23, 1941
- - - July 24, 1941
. . . . Sept. 26, 1941
Op. T. and A. Tu. July 9, 1941

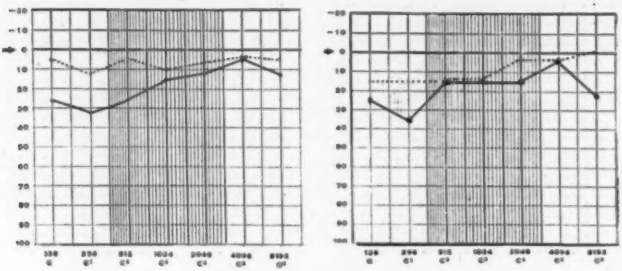
Left
Air Conduction—April 23, 1941
- - - July 24, 1941
. . . . Sept. 26, 1941
Op. T. and A. Tu. July 9, 1941



Case 3. F. K. Age 10.

Right
Air Conduction—Dec. 5, 1939
- - - Jan. 20, 1940
. . . . March 30, 1940
Op. Dec. 21, 1939: A. Tu.

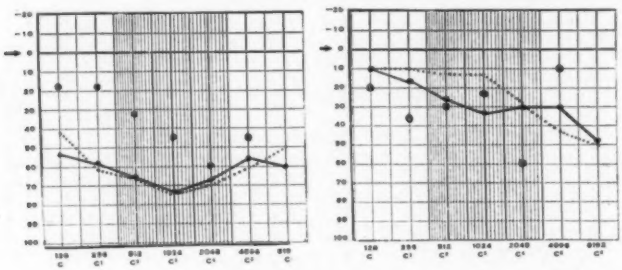
Left
Air Conduction—Dec. 5, 1939
- - - Jan. 20, 1940
. . . March 30, 1940
Op. Dec. 21, 1939: A. Tu.



Case 4. H. S. Age 6½.

Right
Air Conduction—April 8, 1941
. . . . June 14, 1941
Op. April 9, 1941: T and A. Tu.

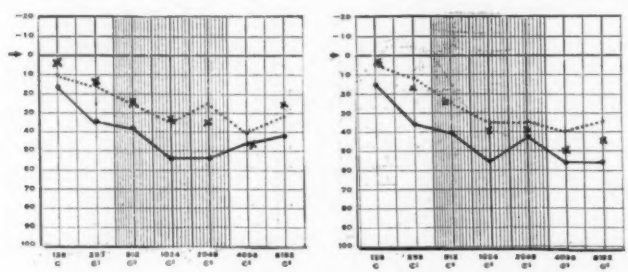
Left
Air Conduction—April 8, 1941
. . . . June 14, 1941
Op. April 9, 1941: T. and A. Tu.



Case 5. J. B. Age 9.

Right
Air Conduction—Oct. 21, 1940
. . . . Sept. 20, 1941
o o o Bone Conduction—Oct. 21, 1940
Op. Jan. 24, 1941: 2nd T. and A. Tu.

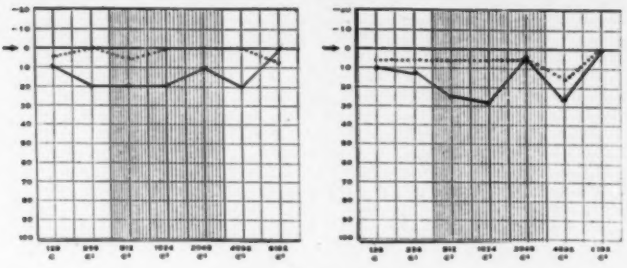
Left
Air Conduction—Oct. 21, 1940
. . . . Sept. 20, 1941
o o o Bone Conduction—Oct. 21, 1940
Op. Jan. 24, 1941: 2nd T. and A. Tu.



Case 6. G. M. Age 12.

Right
Air Conduction—Aug. 8, 1939
. . . . May 27, 1942
x x x Sept. 17, 1942
Op. Sept. 19, 1939: A. Tu

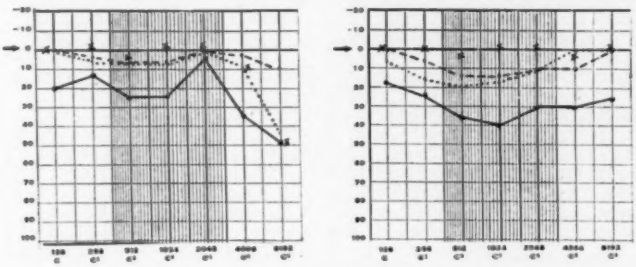
Left
Air Conduction—Aug. 8, 1939
. . . May 27, 1942
x x x Sept. 17. 1942
Op. Sept. 19, 1939: A. Tu.



Case 7. D. B. Age 7.

Right
Air Conduction—Nov. 16, 1939
. . . May 16, 1940
Op. Jan. 5, 1940: T. and A. Tu.

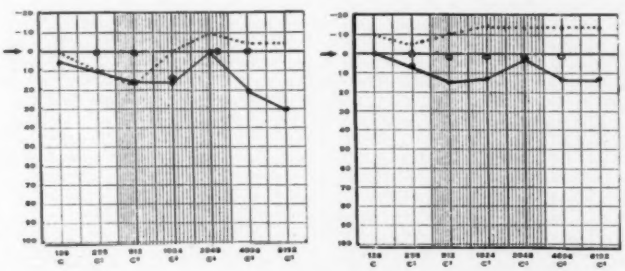
Left
Air Conduction—Nov. 16, 1939
. . . May 16, 1940
Op. Jan. 5, 1940: T. and A. Tu.



Case 8. Di. B. Age 7.

Right
Air Conduction—April 6, 1939
- - - July 6, 1939
. . . . June 13, 1940
x x x March 23, 1941
Op. April 20, 1939: 2nd T. and A. Tu.

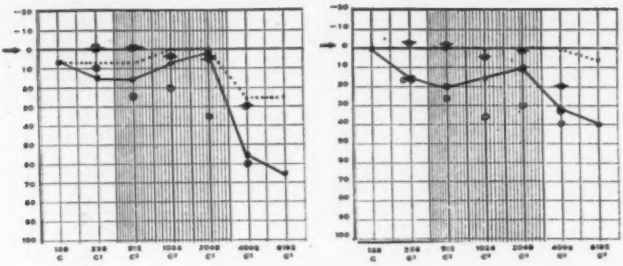
Left
Air Conduction—April 6, 1939
- - - July 6, 1939
. . . . June 13, 1940
x x x March 23, 1941
Op. April 20, 1939: 2nd T. and A. Tu.



Case 9. H. G. Age 7.

Right
Air Conduction—Jan. 5, 1939
. . . . Dec. 7, 1939
o o o Bone Conduction—Jan. 5, 1939
Op. July 14, 1939: A. Tu.

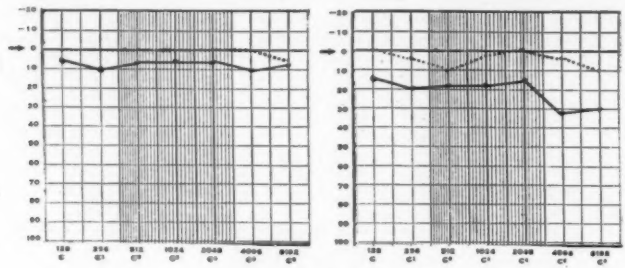
Left
Air Conduction—Jan. 5, 1939
. . . . Dec. 7, 1939
o o o Bone Conduction—Jan. 5, 1939
Op. July 14, 1939: A. Tu.



Case 10. J. H. Age 10.

Right
Air Conduction—July 21, 1938
. . . . March 2, 1939
o o o Bone Conduction—July 21, 1938
O O O Mar. 2, 1939—Bone Conduction
Op. Aug. 16, 1938: 2nd T. and A. Tu.

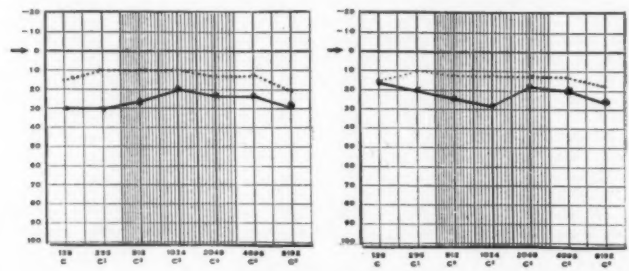
Left
Air Conduction—July 21, 1938
. . . . March 2, 1939
o o o Bone Conduction—July 21, 1938
O O O Mar. 2, 1939—Bone Conduction
Op. Aug. 16, 1938: 2nd T. and A. Tu.



Case 11. A. H. Age 10.

Right
Air Conduction—Nov. 20, 1940
. . . . April 26, 1941
Op. Jan. 24, 1941: 2nd T. and A. Tu.

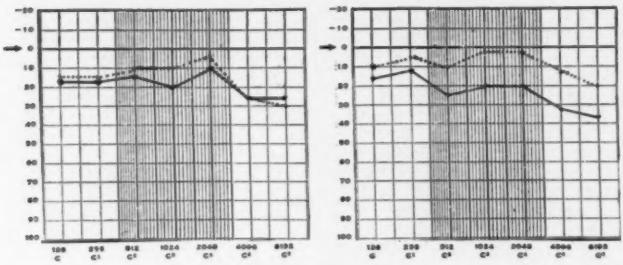
Left
Air Conduction—Nov. 20, 1940
. . . . April 26, 1941
Op. Jan. 24, 1941: 2nd T. and A. Tu.



Case 12. B. H. Age 11.

Right
Air Conduction—March 5, 1941
. . . . April 16, 1941
Op. March 26, 1941: T. and A. Tu.

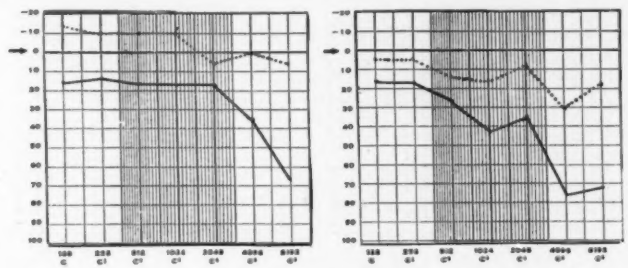
Left
Air Conduction—March 5, 1941
. . . . April 16, 1941
Op. March 26, 1941: T. and A. Tu.



Case 13. R. H. Age 6.

Right
Air Conduction—Feb. 19, 1941
. . . . April 30, 1941
Op. April 23, 1941: T. and A. Tu.

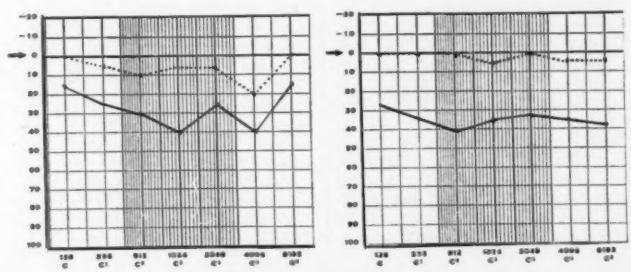
Left
Air Conduction—Feb. 19, 1941
. . . . April 30, 1941
Op. April 23, 1941: T. and A. Tu.



Case 14. M. I. Age 11.

Right
Air Conduction—Oct. 20, 1938
. . . . Jan. 5, 1939
Op. Oct. 28, 1938: T. and A. Tu.

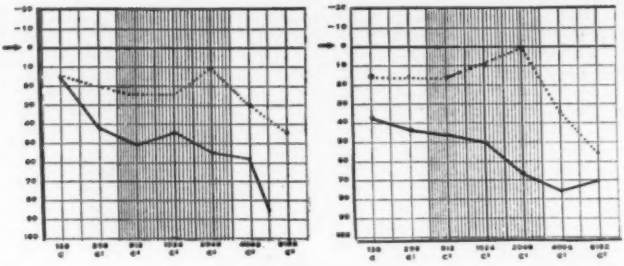
Left
Air Conduction—Oct. 20, 1938
. . . . Jan. 5, 1939
Op. Oct. 28, 1938: T. and A. Tu.



Case 15. P. J. Age 6.

Right
Air Conduction—May 18, 1939
. . . . June 22, 1939
Op. June 9, 1939: T. and A. Tu.

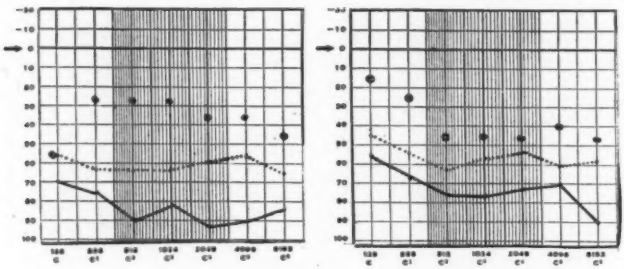
Left
Air Conduction—May 18, 1939
. . . . June 22, 1939
Op. June 9, 1939: T. and A. Tu.



Case 16. B. J. Age 7.

Right
Air Conduction—March 5, 1941
. . . . April 30, 1941
Op. April 23, 1941: A. Tu.

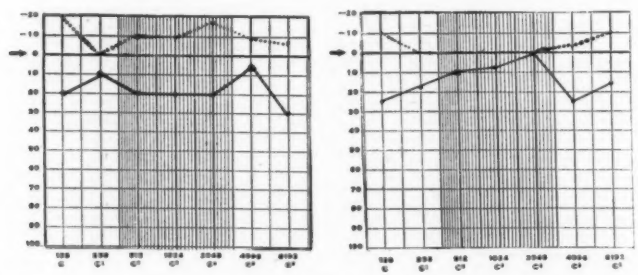
Left
Air Conduction—March 5, 1941
. . . . April 30, 1941
Op. April 23, 1941: A. Tu.



Case 17. G. Mc. Age 6.

Right
Air Conduction—Dec. 4, 1940
. . . . April 30, 1941
o o o Bone Conduction—Dec. 12, 1940
Op. Apr. 23, 1941: A. Tu.

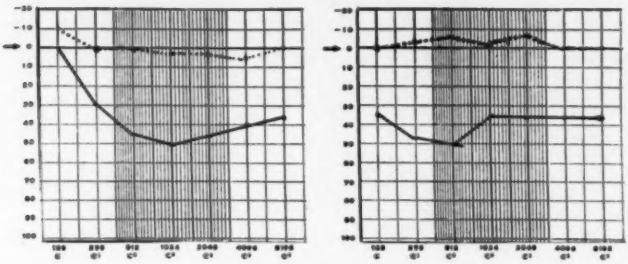
Left
Air Conduction—Dec. 4, 1940
. . . . April 30, 1941
o o o Bone Conduction—Dec. 12, 1940
Op. Apr. 23, 1941: A. Tu.



Case 18. D. M. Age 5½.

Right
Air Conduction—April 20, 1939
. . . . Oct. 26, 1939
Op. May 26, 1939: T. and A. Tu.

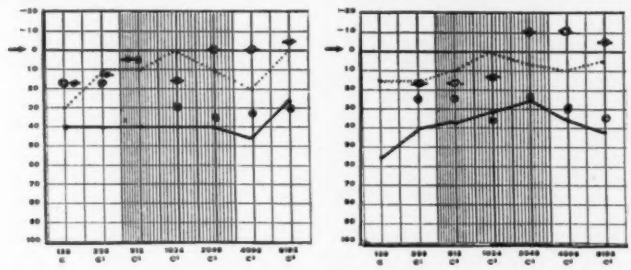
Left
Air Conduction—April 20, 1939
. . . . Oct. 26, 1939
Op. May 26, 1939: T. and A. Tu.



Case 19. C. M. Age 12.

Right
Air Conduction—Nov. 1, 1938
. . . . April 20, 1939
Op. Nov. 4, 1938: A. Tu.

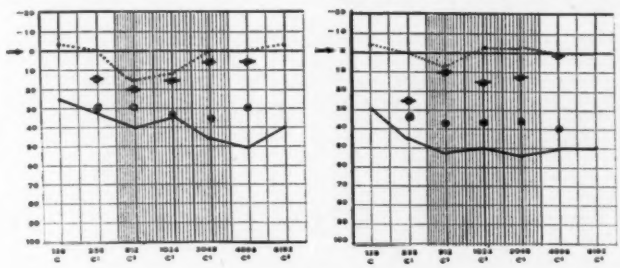
Left
Air Conduction—Nov. 1, 1938
. . . . April 20, 1939
Op. Nov. 4, 1938: A. Tu.



Case 20. Cl. M. Age 5.

Right
Air Conduction March 31, 1939
. . . . July 20, 1939
o o o Bone Conduction—Mar. 1, 1939
O O O Bone Conduction—July 20, 1939
Op. July 14, 1939: T. and A. Tu.

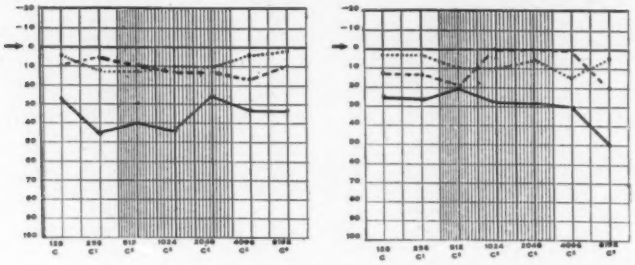
Left
Air Conduction March 31, 1939
. . . . July 20, 1939
o o o Bone Conduction—Mar. 1, 1939
O O O Bone Conduction—July 20, 1939
Op. July 14, 1939: T. and A. Tu.



Case 21. L. M. Age 12.

Right
Air Conduction—Jan. 1, 1939
. . . . July 6, 1939
o o o Bone Conduction—Jan. 1, 1939
O O O Bone Conduction—May 25, 1939
Op. Tu.

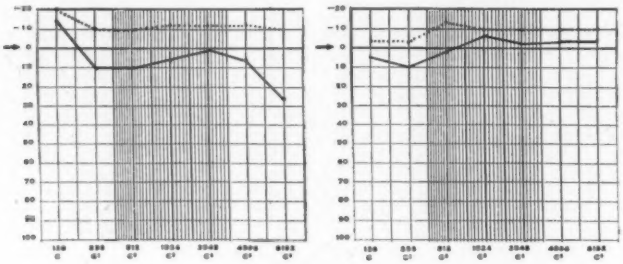
Left
Air Conduction—Jan. 1, 1939
. . . . July 6, 1939
o o o Bone Conduction—Jan. 1, 1939
O O O Bone Conduction—May 25, 1939
Op. Tu.



Case 22. Ge. M. Age 5½.

Right
Air Conduction—Oct. 27, 1938
- - - Nov. 29, 1938
. . . . Jan. 10, 1939
Op. Nov. 17, 1938: Tu.

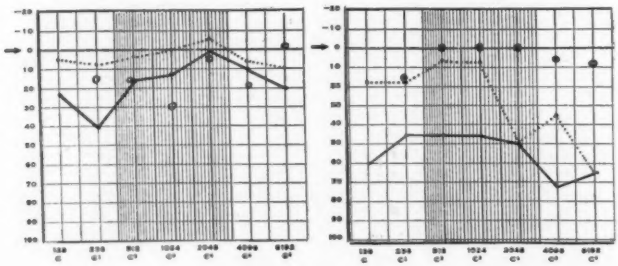
Left
Air Conduction—Oct. 27, 1938
- - - Nov. 29, 1938
. . . . Jan. 10, 1939
Op. Nov. 17, 1938: Tu.



Case 23. C. R. Age 10.

Right
Air Conduction—Oct. 4, 1938
. . . . Nov. 30, 1939
Op. Nov. 4, 1938: T. and A. Tu.

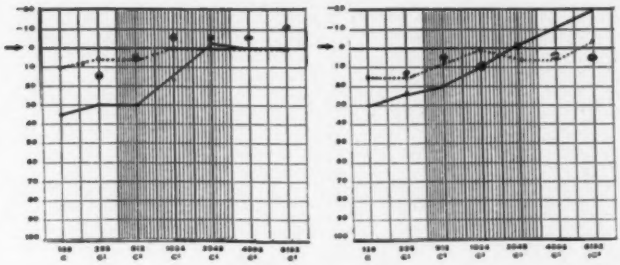
Left
Air Conduction—Oct. 4, 1938
. . . . Nov. 30, 1939
Op. Nov. 4, 1938: T. and A. Tu.



Case 24. J. R. Age 8.

Right
Air Conduction—Jan. 18, 1940
. . . . April 25, 1940
o o o Bone Conduction—Jan. 18, 1940
Op. Feb. 16, 1940: T. and A. Tu.

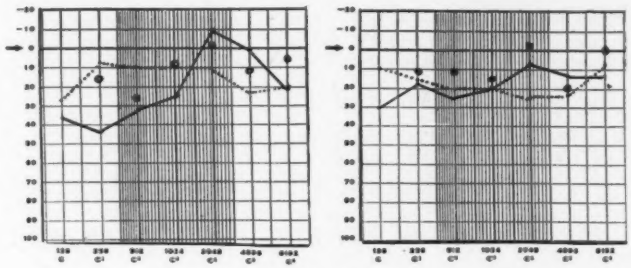
Left
Air Conduction—Jan. 18, 1940
. . . . April 25, 1940
o o o Bone Conduction—Jan. 18, 1940
Op. Feb. 16, 1940: T. and A. Tu.



Case 25. E. S. Age 9.

Right
Air Conduction—April 11, 1940
. . . . July 11, 1940
o o o Bone Conduction—Apr. 11, 1940
Op. April 26, 1940: T. and A. Tu.

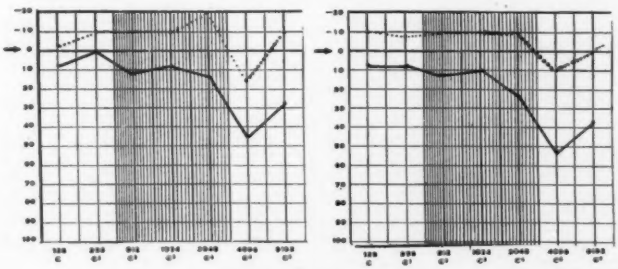
Left
Air Conduction—April 11, 1940
. . . . July 11, 1940
o o o Bone Conduction—Apr. 11, 1940
Op. April 26, 1940: T. and A. Tu.



Case 26. K. S. Age 9.

Right
Air Conduction—Oct. 20, 1938
. . . . Jan. 19, 1939
o o o Bone Conduction—Oct. 20, 1938
Op. Jan. 6, 1939: A. Tu.

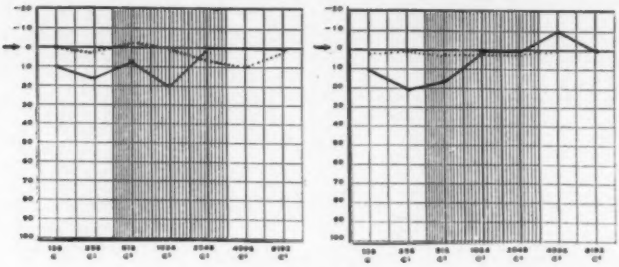
Left
Air Conduction—Oct. 20, 1938
. . . . Jan. 19, 1939
o o o Bone Conduction—Oct. 20, 1938
Op. Jan. 6, 1939: A. Tu.



Case 27. J. S. Age 13.

Right
Air Conduction—March 30, 1939
. . . . Nov. 30, 1939
Op. June 9, 1939: A. Tu.

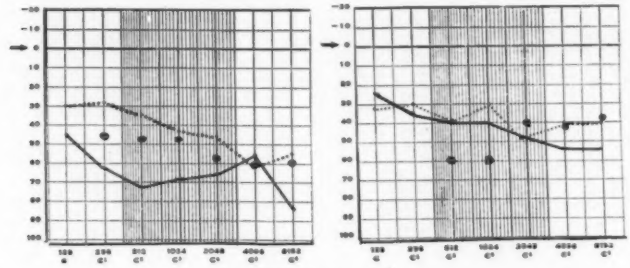
Left
Air Conduction—March 30, 1939
. . . . Nov. 30, 1939
Op. June 9, 1939: A. Tu.



Case 28. R. S. Age 8.

Right
Air Conduction—May 2, 1940
. . . . April 9, 1941
Op. Feb. 26, 1941: 2nd T. and A. Tu.

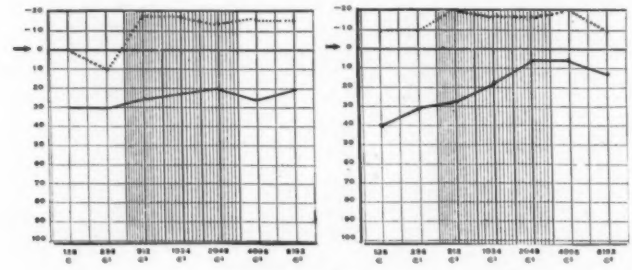
Left
Air Conduction—May 2, 1940
. . . . April 9, 1941
Op. Feb. 26, 1941: 2nd T. and A. Tu.



Case 29. T. S. Age 12.

Right
Air Conduction—Feb. 5, 1941
. . . . April 23, 1941
o o o Bone Conduction—Feb. 12, 1941
No Op.

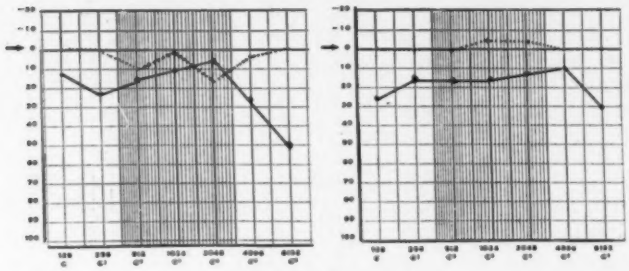
Left
Air Conduction—Feb. 5, 1941
. . . . April 23, 1941
o o o Bone Conduction—Feb. 12, 1941
No Op.



Case 30. V. V. Age 5.

Right
Air Conduction—March 23, 1939
. . . . Nov. 9, 1939
Op. June 9, 1939: 2nd T. Tu.

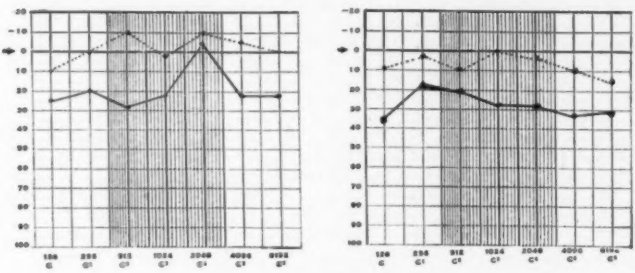
Left
Air Conduction—March 23, 1939
. . . . Nov. 9, 1939
Op. June 9, 1939: 2nd T. Tu.



Case 32. F. Y. Age 10.

Right
Air Conduction—Feb. 9, 1939
. . . . June 7, 1939
Op. March 3, 1939: 2nd T. and A. Tu.

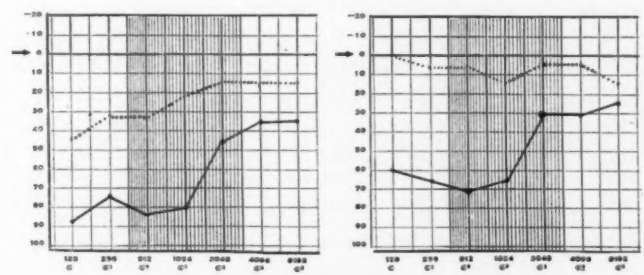
Left
Air Conduction—Feb. 9, 1939
. . . . June 7, 1939
Op. March 3, 1939: 2nd T. and A. Tu.



Case 33. R. B. Age 5.

Right
Air Conduction—May 1, 1942
. . . . July 30, 1942
Op. June 26, 1942: T. and A. Tu.

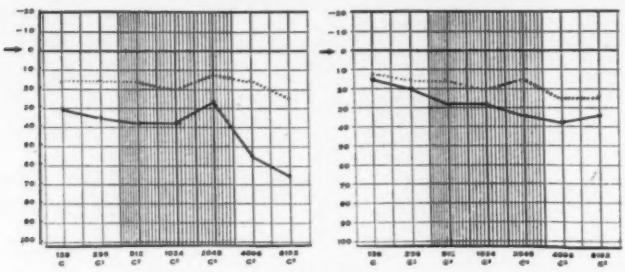
Left
Air Conduction—May 1, 1942
. . . . July 30, 1942
Op. June 26, 1942: T. and A. Tu.



Case 34. B. T. Age 6.

Right
Air Conduction—March 30, 1939
. . . . May 11, 1939
Op. April 29, 1939: T. and A. Tu.

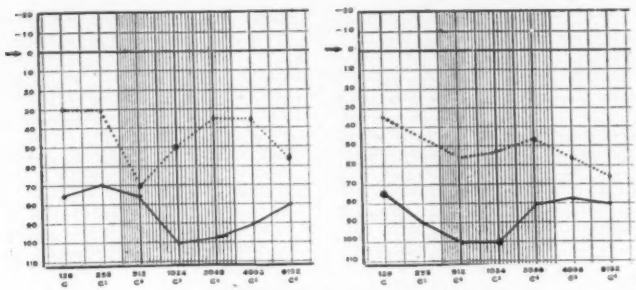
Left
Air Conduction—March 30, 1939
. . . . May 11, 1939
Op. April 29, 1939: T. and A. Tu.



Case 35. W. T. Age 8.

Right
Air Conduction—Feb. 26, 1941
. . . . April 16, 1941
Op. March 20, 1941: T. and A. Tu.

Left
Air Conduction—Feb. 26, 1941
. . . . April 16, 1941
Op. March 20, 1941: T. and A. Tu.



Case 36. L. T. Age 6.

Right
Air Conduction—Jan. 21, 1942
. . . . May 8, 1942
Op. Feb. 20, 1942: T. and A. Tu.

Left
Air Conduction—Jan. 21, 1942
. . . . May 8, 1942
Op. Feb. 20, 1942: T. and A. Tu.

ENDAURAL ATTICOMASTOIDECTOMY: AN EVALUATION.*

DR. FRANK D. LATHROP, Boston.

Otologists are confronted frequently with the problem of what further treatment should be instituted for the patient afflicted with chronic suppurative otitis media presenting a superior marginal or attic perforation and a foul discharge, and in whom the involved ear retains serviceable hearing but has failed to respond to adequate medical treatment. On such occasions a decision must be made either to continue medical care or to advise surgical intervention.

If medical treatment is continued, both the otologist and the patient should realize that in all probability the otorrhea will persist, and further impairment of hearing may occur in spite of judicious use of astringent or antiseptic ear drops, attic irrigations, dry wipes or dusting powders, and removal of granulation tissue either by excision or chemical cautery. Furthermore, since the tympanic membrane presents either attic or marginal perforation, pseudo-cholesteatomatous deposit or bone necrosis in the middle ear, epitympanic space or mastoid process is frequently present and may progress in spite of an adequate medical regimen. Progression of these pathologic processes may lead to complications that require surgical intervention at a less opportune time and result in further decrease of auditory acuity.

Surgical intervention, on the other hand, should be so planned that in the majority of cases cessation of the chronic otorrhea is obtained and the preoperative hearing level either is maintained or improved; also, surgical removal of the cause of the existing disease should eliminate the danger of extension of the infectious process beyond the confines of the temporal bone. Endaural atticomastoidectomy, as I shall describe it, satisfies these requirements.

For many years the development of a satisfactory endaural approach to the mastoid process has intrigued otologists.

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*Presented as Candidate's Thesis for the American Laryngological, Rhinological and Otological Society, 1943.

Thies, in 1907, is credited with first performing a radical mastoidectomy through the external auditory canal. Since then, interest in similar approaches to the mastoid process has risen and fallen as various methods have been introduced and later proven unsatisfactory. In recent years, however, the endaural techniques of Guns and Lempert have recreated interest in these methods of approach to the mastoid process of the temporal bone.

OPERATIVE PROCEDURE.

Lempert designated the area of soft tissue of the ear bounded posteriorly by the lower end of the helix and anteriorly by the upper part of the tragus as the antauricular

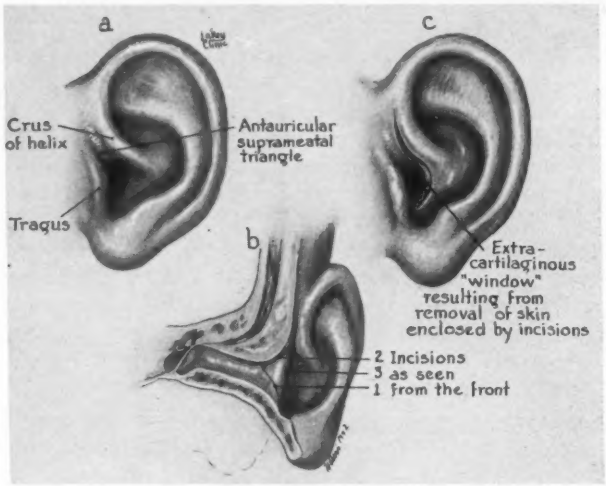

Fig. 1.

suprameatal membranous triangle and established it as the anatomic basis for the formation of the endaural approach (see Fig. 1a). The mobile membranous and extracartilaginous endaural approach for a modified radical mastoidectomy is created by three incisions (see Fig. 1b). The first incision begins in the posterior superior canal wall of the external auditory canal at the junction of the membranous and osseous portions and is carried downward and laterally through the membranous wall to the lower end of the anterior border of

the concha. The second incision begins where the first one started and is carried outward along the superior posterior wall of the canal to the base of the previously described antauricular suprameatal membranous triangle. From this point the incision is extended along the anterior border of the triangle to its apex, where the helix and tragus almost meet. The third incision connects the outer ends of the two previous incisions along the anterior border of the concha.

These incisions are carried down to the bone, and the enclosed triangular piece of soft tissue is removed by means of a subperiosteal elevator and scissors (see Fig. 1c). The postauricular skin and periosteum covering the mastoid process, together with the antauricular skin and periosteum covering the root of the zygoma, are then elevated. The self-retaining retractor is inserted and spread, producing a mem-

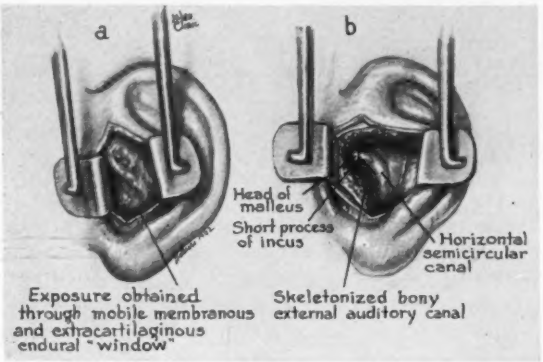

Fig. 2.

branous, extracartilaginous, mobile, endaural window which exposes to view all parts of the mastoid process and tympanic cavities (see Fig. 2a).

Through this approach, the mastoid cortex is removed with gouge and mallet, and the mastoid antrum opened as through the postauricular approach. Beginning with the antrum, all granulation tissue, cholesteatomatous material and diseased bone are removed by means of gouge and mallet, burrs and curettes, until Troutman's triangle, the sigmoid plate, the horizontal semicircular canal and the floor of the antrum are

well delineated, the posterior bony canal wall and mastoid tegmen skeletonized, and the overhanging cortex removed. It is advisable, however, not to remove more of the eburnated bone of the mastoid process than is necessary to eradicate the disease. The lateral wall of the epitympanic space is then removed with sharp curettes, gouge and mallet until the short process of the incus, the head of the malleus and the incudomalleal joint are well visualized (see Fig. 2b). The removal of bone is carried forward into the posterior root of the zygoma to the junction of the superior and anterior walls of the bony external auditory canal.

With sharp curettes the posterior and superior bony walls of the external auditory canal are skeletonized further to paper thinness. During this procedure care should be exercised not to fracture the bone, as a tear in the cutaneous lining of the external auditory canal may occur and make the formation of a plastic flap difficult. Then the bony external auditory canal wall is carefully resected away from the cutaneous lining of the canal with rongeurs to the level of the facial ridge posteriorly and the notch of Rivinus superiorly. An excellent view of the epitympanic space and its contents can now be obtained.

The suspensory ligament of the malleus and the incudomalleal articulation are disrupted with a paracentesis knife and the incus removed, care being exerted not to avulse the footplate of the stapes from its seat within the oval window. Amputation of the head of the malleus is performed with a small clipper such as is used to excise the cuticle about the fingernail. All pathologic tissue is removed from the epitympanic space and the tegmen tympani skeletonized until it blends with the tegmen antri.

Upon completion of careful toilet of the epitympanic space, the facial ridge is carried down to the lowest limits of safety with respect to the facial nerve and smoothed with either sharp curettes or burrs. The membrana tensa is freed from the annulus in its posterior superior aspect, allowing slight mobilization of the tympanic membrane in this area and partial mobilization of the interior of the middle ear (see Fig. 3a). Through this increased exposure of the interior of the middle ear and the pre-existing perforation in the tympanic

membrane, any granulation tissue or cholesteatomatous deposit is removed with middle ear forceps before attention is turned to the formation of the plastic flap.

The plastic flap is formed from the cutaneous lining of the posterior, superior and anterosuperior walls of the external auditory canal and the attached tympanic membrane. The first incision begins at the junction of the superior and anterior canal walls and is carried inferiorly and medially to half the depth of the anterior canal wall. At this point the incision is continued medially but superiorly so that it ends at

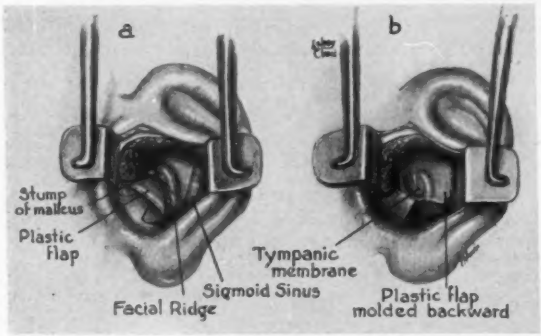

Fig. 3.

the annulus at the level of the anterior malleolar fold. The second incision begins at the junction of the posterior and inferior canal walls and extends medially and slightly anteriorly to half the depth of the posterior canal wall. At this point it bends posteriorly so that it ends at the annulus in the region of the junction of the inferior and posterior quadrants of the tympanic membrane. The posterior and superior portions of the cutaneous canal wall, bounded by these two incisions, are elevated from the bony attachments with a sharp submucous elevator until mobilized to the level of the annulus. The plastic flap is then molded backward over the facial ridge so that the medial wall of the epitympanic space is covered by the remaining portion of the membrana flaccida while the cutaneous portion covers the facial ridge and basal labyrinthine portion of the petrous pyramid (see Fig. 3b). The bony anterosuperior canal wall is smoothed with curettes, and the plastic flap held in place with paressine

mesh gauze packs. The self-retaining retractor is removed and the skin incisions allowed to fall together spontaneously. The usual outside mastoid head dressing is applied.

The postoperative care is simple. The mastoid packing is removed on the fifth or sixth postoperative day, and the outside head dressing omitted if so desired. The operative cavity is not repacked. Thereafter, the mastoid cavity and middle ear are kept clean of secretions, dusted with sulfanilamide or sulfathiazole powder, and the skin incision in the suprameatal membranous triangle carefully spread to obviate atresia or stenosis of the conchal opening. The conchal opening is protected with sterile cotton until excessive secretion ceases. The formation of exuberant granulation tissue is controlled by the judicious application of 70 per cent silver nitrate. Complete healing usually occurs within two or three months.

RESULTS IN 15 CASES.

Since September, 1940, I have performed 15 endaural modified radical mastoidectomies for chronic suppurative otitis media. These infections were of the dangerous type and had failed to respond to adequate medical treatment. In the majority of cases chronic otorrhea had been present either constantly or intermittently for many years. Only four of the patients gave a history of chronic suppurative otitis media of less than three years' duration. In all cases the discharge was purulent, and in 10 the odor of the discharge was characteristic of necrosis of the bone or cholesteatoma. The presence of necrosis, cholesteatoma or both was confirmed at operation in eight of the 10 cases.

In all cases the perforation was located either in the membrana flaccida or involved both the membrana flaccida and tensa. Whenever the membrana tensa was involved the perforation was a marginal one and was associated with destruction of the annulus. Two patients had had a previous simple mastoidectomy performed elsewhere, followed by revisions. One of these developed a postauricular fistula following the second revision. The history and clinical data are presented in Table 1.

Twelve (80 per cent) of the 15 ears operated upon are now dry. The operation was a failure in three cases, in which

TABLE 1. HISTORY AND CLINICAL DATA.

Case No.	Age in Yrs.	History	Location and Type of Perforation (Operated Ear)
1	28	Intermittent, malodorous, mucopurulent discharge for 7 years.	Large posterosuperior perforation involving membrana flaccidda.
2	18	Intermittent, foul, purulent discharge for 12 years.	Posterosuperior perforation of moderate size in membrana flaccida and tensa.
3	20	Intermittent, malodorous, mucopuruient discharge bilaterally for as long as he can remember.	Large posterosuperior marginal perforation involving Shrapnell's area.
4	19	Simple mastoidectomy, 8 years of age; revision, 10 years of age; ear dry for 7 years; constant, foul, purulent discharge for last 2 years.	Moderate-sized anterosuperior perforation in membrana flaccida with extension into membrana tensa.
5	18	Almost constant, foul, purulent discharge bilaterally for 10 years.	Large posterosuperior perforation in membrana flaccida with granulation tissue on posterior canal wall at annulus.
6	18	Almost constant, foul, purulent discharge bilaterally for 10 years.	Large posterosuperior perforation involving both membrana tensa and flaccida.
7	19	Constant, malodorous, purulent discharge for 15 months.	Large perforation in membrana flaccida with epithelial debris in attic.
8	22	Intermittent pain in ear with associated malodorous, purulent discharge for years.	Large posterosuperior perforation involving membrana tensa and flaccida with polyp attached to annulus.
9	17	Constant, foul, purulent discharge for 5 years following scarlet fever.	Large perforation in membrana tensa with partial destruction of scutum.
10	14	Constant, foul, purulent discharge bilaterally for 2 years.	Moderate-sized anterosuperior perforation in membrana flaccida.
11	22	Constant, malodorous, purulent discharge for as long as he can remember.	Moderate-sized anterosuperior perforation involving membrana tensa and flaccida at annulus.
12	51	Simple mastoidectomy at 7 years, followed by revisions at age of 15 years and 32 years, which failed to relieve pain, vertigo or foul discharge.	Large posterosuperior perforation in membrana flaccida.
13	40	Intermittent, foul, purulent discharge for 30 years.	Moderate-sized perforation in membrana flaccida with epithelial debris in attic.
14	33	Constant, foul, purulent discharge for as long as he can remember.	Large posterosuperior perforation involving membrana tensa and flaccida.
15	33	Constant, foul, purulent discharge for 2½ years.	Large perforation in membrana flaccida with partial destruction of scutum.

the chronic otorrhea persists but in decreased amount. Complicating factors are present in these three patients, however, which may have influenced the final result. One patient had uncontrolled chronic nephritis of moderate degree. In this case the ear continues to discharge a small amount of mucus intermittently; this is not noticed by the patient but collects in the operative cavities as crusts. Another patient went swimming before the middle ear was entirely dry, with the result that the ear has continued to discharge moderately. This patient also has a large pad of infected adenoid tissue about the Eustachian tube orifice of the discharging ear, for which operation has been advised. The third failure is complicated by the fact that during the operation the cutaneous plastic flap together with the remaining portion of the membrana flaccida was accidentally avulsed with the result that an opening of considerable depth now exists between the superior border of the membrana tensa and the medial wall of the middle ear. This opening has never entirely closed and intermittently the ear discharges a small amount of mucopus. Information concerning the operative findings, complications, postoperative convalescence and end-results of all cases is presented in Table 2.

The auditory acuity was either improved or remained unchanged in 13 of the 15 ears upon which an endaural modified radical mastoidectomy was performed. In the remaining two cases the impairment in hearing was worse following operation. An improvement or further impairment of auditory acuity was not considered present unless the audiogram, obtained when the ear was entirely healed, revealed an average gain or loss for the critical frequencies of 512, 1,024 and 2,048 greater than 10 dcb. as compared with the same frequencies of the preoperative audiogram.

Six (40 per cent) of the 15 patients obtained an increase in auditory acuity. Four of these six patients revealed improvement in hearing ranging from 12 to 21 dcb. as determined by comparison of the preoperative and postoperative audiograms. Two of the patients were admitted directly to the hospital and, consequently, preoperative audiograms were not obtained; however, residual voice tests revealed the hearing to be decreased to 6/20 and 5/20 preoperatively and increased postoperatively to 15/20 and 18/20 respectively.

TABLE 2. OPERATIVE FINDINGS AND END-RESULTS.

Case No.	Operative Findings	Operative Complications	Post-operative Convalescence	End Result*
1	Sclerotic mastoid with granulation tissue in periantral cells, antrum, attic and middle ear. Incus necrotic.	None	Uneventful	Ear dry. Hearing improved.
2	Sclerotic mastoid with antral enlargement. Cholesteatoma in antrum, attic and middle ear. Incus necrotic and disconnected from malleus.	None	Uneventful	Ear dry. Hearing unchanged.
3	Diploic and sclerotic mastoid. Granulation tissue in antrum, attic and middle ear. Incus and head of malleus necrotic.	None	Uneventful	Ear dry. Hearing unchanged.
4	Sclerotic mastoid with surgical defect in antral region. Granulation tissue in antrum and attic. Polyp in middle ear. Head of malleus necrotic.	None	Uneventful	Ear dry. Hearing improved.
5	Sclerotic mastoid with antral enlargement. Granulation tissue in antrum and attic. Head of malleus necrotic.	None	Uneventful	Ear dry. Hearing improved.
6	Sclerotic mastoid with antral enlargement. Granulation tissue in antrum and attic.	None	Uneventful	Ear dry. Hearing unchanged.
7	Marked necrosis of incus. Head of malleus absent. Cholesteatoma and granulation tissue in antrum, attic and middle ear.	None	Uneventful	Ear dry. Hearing improved.
8	Sclerotic mastoid with antral enlargement. Granulation tissue and cholesteatoma in antrum, attic and middle ear. Incus and head of malleus necrotic.	None	Uneventful	Occasional discharge. Hearing worse.
9	Sclerotic mastoid. Cholesteatoma in antrum and attic. Necrosis of aditus, incus and head of malleus.	None	Uneventful	Ear dry. Hearing improved.
10	Diploic mastoid. Granulation tissue in antrum and attic.	None	Uneventful	Ear discharging less. Hearing worse.
11	Sclerotic mastoid with antral enlargement. Granulation tissue in antrum and attic. Head of malleus necrotic.	None	Uneventful	Ear dry. Hearing unchanged.
12	Sclerotic mastoid with surgical defect in antral region. Postauricular fistula. Granulation tissue and cholesteatoma in antrum and attic. Incus absent. Head of malleus necrotic.	None	Uneventful	Ear dry. Hearing improved.
13	Sclerotic mastoid with antral enlargement. Incus and head of malleus absent. Granulation tissue in antrum and attic.	None	Uneventful	Ear dry. Hearing unchanged.
14	Sclerotic mastoid with antral enlargement. Granulation tissue and cholesteatoma in antrum and attic. Incus absent and head of malleus necrotic.	None	Uneventful	Ear dry. Hearing unchanged.
15	Cholesteatoma and foul debris in antrum and attic. Incus necrotic.	Cutaneous plastic flap and membrana flaccida avulsed	Uneventful	Ear discharging less. Hearing unchanged.

*Hearing not considered improved or made worse unless average decibel gain or loss for above frequencies was greater than ±10 dcb. or equivalent for residual voice.

The improvement in hearing was unchanged in seven and worse in two cases postoperatively. The average decibel loss for the critical frequencies in the latter two cases was 13 dbs.; however, neither patient noted a subjective increase in deafness and both are included in the group of three who failed to obtain a dry ear.

The data relative to the effect of endaural atticomastoidectomy upon the auditory acuity is presented in Table 3. Postoperatively, Case 10 presented a hearing loss of 17 dcb.

TABLE 3. AVERAGE DECIBEL LOSS FOR CONVERSATIONAL FREQUENCIES 512, 1,024, 2,048.

Case No.	Preoperative		Postoperative		Postoperative Average Gain or Loss (Oper. Ear)
	Oper. Ear	Unop. Ear	Oper. Ear	Unoper. Ear	
1	35	47	23	48	+12
2	30	0	27	0	+ 3
3	57	37	50	30	+ 7
4	38	0	18	0	+20
5	20	18	8	18	+12
6	18	8	18	8	0
7	43	0	22	0	+21
8	27	0	40	0	—13
9	6/20*	20/20*	15/20*	20/20*	+9/20*
10	20	0	33	17	—13
11	27	25	32	17	— 5
12	5/20*	18/20*	12/20*	18/20*	+7/20*
13	33	0	25	0	+ 8
14	25	0	22	0	+ 3
15	22	17	22	20	0

*Residual voice. Patient admitted directly to hospital so that audiometric studies could not be obtained.

in the unoperated ear, whereas, postoperatively, the hearing for the conversational frequencies was normal. This is explained by the fact that this patient had bilateral chronic suppurative otitis media.

CONCLUSIONS.

Endaural atticomastoidectomy should be reserved for those patients with chronic suppurative otitis media presenting a superior marginal or attic perforation, in whom the condition fails to respond to adequate medical treatment, and who retain serviceable hearing in the involved ear. Furthermore, carefully selected patients afflicted with bilateral chronic suppurative otitis media whose hearing loss bilaterally is of such degree that it is difficult for them to converse with ease may

have benefit in hearing by an endaural modified radical mastoidectomy on one ear, providing the bone conduction is good. In two patients in whom operation was performed with this idea in mind, one (Case 1) was distinctly benefited, while the other (Case 3) was not.

The endaural rather than the postauricular approach to the temporal bone is preferred in this type of modified radical mastoidectomy because the exposure affords greater accessibility to the epitympanic space and facilitates the removal of all disease tissue from this region. Furthermore, the plastic flap is continuous with the tympanic membrane and may aid in the formation of scar tissues to close the perforation in the tympanic membrane. The excision of the triangular piece of skin contained between the endaural incisions removes, to a large degree, the ceruminal and sebaceous glands in the membranous portion of the cutaneous lining of the external auditory canal and allows a cleaner operative field, requiring less attention after the cavity is healed.

The removal of the incus and the amputation of the head of the malleus, which results in disruption of the ossicular chain, may appear to many otologists to be a radical and unnecessary procedure; however, this is an important step in the performance of an adequate modified radical mastoidectomy since by no other means can one remove all the pathologic tissue within the epitympanic space and stop the discharge. Furthermore, in all but two of my cases there was necrosis of the incus, the head of the malleus or both the incus and the malleus. This necessitated removal of the necrotic portions of the ossicular chain in order to eradicate all disease of the epitympanic space.

Kopetzky, at the International Otological Congress held in Boston in 1912, criticized the modified radical mastoidectomy as described by Heath and advocated by Bryant. He stated that it failed to meet any of the requirements of radical mastoid surgery; that in chronic suppurative otitis media of long duration it could not restore to functional activity a necrotic ossicular chain or heal areas of necrosis in the annulus or tympani, adital and antral walls, and that if a simple mastoidectomy were performed in the same type of case for which Heath and Bryant advocated a modified radical mas-

toidectomy, the result as far as hearing was concerned would be as good.

I agree with Kopetzky's criticisms of the Heath operation, for in this procedure the epitympanic space is not opened widely and the pathologic tissue contained therein carefully removed; however, I do not believe that these criticisms hold true for the endaural modified radical mastoidectomy. The objectives of a radical mastoidectomy are to eradicate the disease, prevent extension beyond the confines of the temporal bone and cause cessation of the chronic otorrhea. The first two objectives were obtained in every case in my series, while cessation of the chronic discharge was obtained in 12 of the 15 cases.

Since the incus and head of the malleus are intentionally removed in order to facilitate painstaking removal of all pathologic tissue from the epitympanic space, endaural atticomastoidectomy will not allow the functional restoration of a diseased ossicular chain; however, necrotic areas of the annulus or tympanic adital and antral walls can be and are removed during the course of this operative procedure.

Six of the 15 ears upon which an endaural modified radical mastoidectomy was performed obtained definite auditory improvement, seven remained unchanged, and in two hearing was further reduced. I rather doubt that if a simple mastoidectomy had been performed in these 15 cases the effects upon the hearing would have been as beneficial. It is also extremely doubtful that a simple mastoidectomy would have effected a dry ear in any of these cases, and continuation of the chronic otorrhea would have allowed a decrease in auditory acuity. This is borne out by the fact that of three cases in which otorrhea persisted, two exhibited further impairment of hearing.

Woodruff and Henner have recently reported their experiences with endaural atticomastoidectomy performed in a manner similar to that which I have described. Cessation of the chronic otorrhea was obtained in four of their five cases. An improvement in hearing for the spoken or whispered voice was obtained in three of the five cases. In their opinion this operation in selected cases permits the complete removal of

disease tissue from the mastoid and epitympanic space and the preservation or even improvement of hearing.

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CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1944 for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the Secretary, Dr. Arthur W. Proetz, 1010 Beaumont Building, St. Louis 8, Mo., before March 1, 1944.

NEUROLOGY IN OTOLARYNGOLOGY.*

DR. HANS BRUNNER, Chicago.

NEUROLOGY OF LARYNX.

Paralysis of the Larynx: Suehs[18] analyzes the records of 270 cases of vocal cord paralysis which have accumulated over a period of 18 years. He believes that the presence or absence of tension of the paralyzed cord is the best index to the degree of paralysis. In other words, if tension is lost and the cord is fixed, he considers the paralysis as being complete, regardless of whether the corresponding arytenoid is in the median line or cadaveric position. Unfortunately, he does not say by which method he measures the tension of the vocal cord.

The immobile vocal cord was more often in the median line than in the cadaveric position in his series. The voice cannot be relied upon as a criterion for the presence or absence of paralysis of the vocal cord, because when the tension of the cord remains good, the voice is often normal. This occurs even in bilateral abductor paralysis.

Carcinoma is the most frequent single cause of unilateral paralysis. Carcinoma of the esophagus was the most frequent tumor causing paralysis, and following in order of frequency were tumors of the lung and/or mediastinum, bronchogenic carcinoma, carcinoma of the pharynx, carcinoma of the trachea and cervical nodes. Thyroidectomy was the most common cause of bilateral abductor paralysis. In Suehs' series preoperative paralysis was exceedingly rare in benign enlargement of the thyroid gland. The left recurrent nerve is seemingly more susceptible to injury than the right, even when intrathoracic diseases are excluded.

Whatever the cause of paralysis of the cord, the prognosis for recovery is poor, but is best in those cases where definite

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*Not all papers mentioned in the bibliography are reviewed in this report.

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etiologic factor can be demonstrated. The etiology often remains obscure in spite of thorough investigation.

Shea[17] reports two cases of paralysis of the right vocal cord due to an aneurysm of the right subclavian artery.

McCall and Gardiner[13] advise a modification of the operation of Kelly which primarily concerns the identification and fixation of the arytenoid cartilage. To overcome the uncertainty as to where to make the window in the thyroid ala, an anterior commissurescope with a bright light is introduced into the larynx in the usual manner as soon as the thyroid alae are exposed. The arytenoid is then engaged by the end of the laryngoscope and firmly pressed laterally against the thyroid alae. The room is then darkened, and it is an easy matter to see just where the window in the thyroid alae should be made. After the window is made, the laryngoscope is reintroduced in the larynx. A small, full-curved atraumatic needle is fixed in the anterior portion of the arytenoid at the cord attachment. The interarytenoid muscle is then severed and the arytenoid cartilage is mobilized by disarticulating it from the cricoid. The end of the suture is then fed through the window and traction on the suture delivers the arytenoid into the window. After the arytenoid is delivered it should be anchored securely with 0.1 chromic gut. Arytenoidectomy is not necessary.

This procedure was perfected in the anatomical laboratory. It seems to the reviewer that it probably will offer some difficulties in actual operations.

Wright[21] has performed the Kelly operation for restoration of laryngeal function following bilateral paralysis of the vocal cord in three females in their late thirties or early forties. Arytenoidectomy was performed on the right side in two cases and on the left side in the other. The postoperative course was normal in all cases.

The first patient obtained noticeable improvement in breathing after a few weeks, but reached maximum improvement only after four months; however, she has not yet been decannulated for psychological reasons as she has kept her tracheotomy tube occluded constantly, except when asleep, for the past four months. Her voice is excellent, though slightly subnormal in clarity.

In the second patient there was a persistent edema of the laryngeal vestibule. This gradually subsided, but eight months after the operation it was impossible to visualize the vocal cords by indirect laryngoscopy. There was similar prolonged edema of the larynx following tracheotomy and some disturbance of the laryngeal lymphatics was evident.

In the third case the vocal cord was sutured to the external perichondrium, creating a permanent glottic space of about 7 mm. Adequate breathing space was obtained and a good speaking voice maintained. The patient was decannulated one month following operation.

Disturbances of Voice: Clerf and Braceland[3] emphasize that the former division of illnesses into the categories, functional versus organic, can no longer be held, for we now realize that all disease processes have both psychic and organic components. While we agree that functional symptoms can be superimposed upon organic diseases, it is obvious that an illness cannot, at the same time, be both functional and organic.

Psychophonasthenia is a partial defect, a way station as it were, on the road to hysterical aphonia which might be considered the end of the line. The trouble underlying the functional aphonias usually originates in an incompletely recognized conflict between the sense of duty, the desire for human respect and the overwhelming desire to escape from difficult situations.

Whenever we encounter hysterical aphonia we should ask ourselves what difficulty is being solved in this temporary and unsatisfactory manner. Our task is to determine how the aphonia is getting the patient out of his difficulty. The successful solution of this problem is the first step in the treatment of the patient.

Mirror laryngoscopy commonly reveals a normal larynx. Deep inspiration will be followed by slight abduction of both cords. On asking the patient to say ah-ah or eh-eh, the cords often meet momentarily in the midline and then separate so that as air is forced between the vocal cords, practically no sound is produced. To ascertain if the cords can meet in the midline the patient is asked to cough with mirror held in the

pharynx. It will be noted that the vocal cords meet normally in the midline.

Malingering must not be confused with hysterical aphonia. The hysteric is quite indifferent and will co-operate very well. This is in contrast to the malingerer who is not anxious to have his larynx examined.

We should never make a diagnosis of functional disease or neurosis by exclusion. In addition to absence of organic or inflammatory disease we must have some positive signs of neurosis.

So far as the treatment is concerned, any unnecessary manipulation or any form of meddlesome surgery simply fixes the neurosis and causes the patient to concentrate his attention upon the part being treated. The laryngologist should rather recognize the essentially psychiatric foundation of these conditions and should either equip himself to deal with them or see that the patient is placed under expert psychiatric treatment.

Goldman and Salmon[8] report 23 cases that manifested striking vocal and laryngeal changes as a result of the intramuscular administration of testosterone propionate. The total amount which these patients received varied from 225 to 3,000 mg. over periods extending from one to 22 months, given in individual doses of 25 or 50 mg. two or three times weekly. The vocal complaints were hoarseness, huskiness, voice with masculine characteristics, constricting or tightening feeling about the larynx. The voices of all these patients fatigued easily.

The vocal cords presented a pale grayish color which became off-white as the process progressed, and they were swollen and enlarged. Caudal extension of the edema in the depth of the vocal cords was often noted. As a result, the subglottic surfaces of these vocal cords were visible. Imperfect approximation of the posterior portions of the vocal cords was also seen in many of these cases. The appearance of the larynx resembled the picture seen in allergic conditions.

In some instances vocal disturbances as well as physical alterations of the vocal cords persisted after discontinuation of the androgen therapy. In other cases a change in the voice

continued after regression of the swelling and congestion of the vocal cords. The latter did not return to a completely normal state but acquired a thickened and pale appearance.

It seems that the vocal and laryngeal changes which occur in females who receive large doses of testosterone propionate are similar to those which develop in boys at puberty. With 200 mg. of testosterone propionate per month the risk of vocal and laryngeal changes is negligible. The threshold for these vocal and laryngeal phenomena is approximately 500 mg. of testosterone propionate per month.

NEUROLOGY OF PHARYNX.

Cricopharyngeal Spasm: Newer radiographic observations support the endoscopic opinions that the cricopharyngeus muscle possesses the function of localized contraction and is in every sense a true sphincter. By using barium or air as contrast-medium, Templeton and Kredel[12] were able to demonstrate the cricopharyngeus muscle at Roentgenologic examination. The air can be forced into the hypopharynx if the patient performs Valsalva's test. This procedure was first described by Joenssen in 1934 and later used for the diagnosis of hypopharyngeal tumors by Waldapfel.

Extensive studies by means of the described technique prove that the force which propels a bolus through the laryngeal pharynx is produced by a peristaltic wave beginning with a diffuse contraction of the oropharynx and progressing in an unbroken manner through the laryngeal pharynx, past the cricopharyngeus muscle and down the esophagus. This wave is preceded by a wave of inhibition which allows the lumen ahead of the wave of contraction to be distended by the bolus. There is no need to postulate an active expansion of the lumen creating a negative pressure which sucks the bolus along.

The cricopharyngeus sphincter is normally closed. The term "closed" does not necessarily connote active muscular contraction. It is supposed that the cricopharyngeus sphincter is held closed against changes in pressure occurring in the pharynx during respiration, coughing, blowing, etc., by the normal tone, probably augmented by additional contraction stimulated by certain nervous reflexes.

In normal individuals the force exerted on the bolus by the peristaltic wave is great enough to overcome any resistance the sphincter offers. As a consequence, passive opening of the sphincter occurs; therefore, any marked weakness in the pharyngeal musculature will fail to force a bolus through a normally closed sphincter. From Roentgenologic observations the term "cricopharyngeal spasm" should not be used, since it is not possible to determine the amount of pressure set up in a pharynx in a patient whose cricopharyngeus muscle fails to open adequately.

Whether abnormal contraction of the cricopharyngeus sufficient to resist the powerful pharyngeal contractions of a normal pharynx exists is not known. In patients suffering from "globus hystericus" abnormal action of the pharyngeal muscle and of the sphincter was not observed.

Nasopharyngeal Tumors: For the early diagnosis of nasopharyngeal tumors Hubert[9] advises the instillation of epinephrine hydrochloride into the conjunctival sac. Loewi has shown that when epinephrine hydrochloride is instilled into the conjunctival sacs of normal persons no mydriasis will ensue; however, if the sympathetic nervous system is rendered hyperirritable, as may occur in persons with exophthalmic goiter, diabetes or nasopharyngeal tumors, dilatation of the pupils will become apparent. In performing this test, one must be patient to wait for the reaction to occur. It may take an hour or longer, depending upon the color of the iris. The darker the color of the iris the longer it takes to get the reaction.

The test shows with a reasonable degree of certainty whether the lesion is *1.* in the superior cervical ganglion and its postganglionic fibres or *2.* in the preganglionic fibres and the central nervous system. In the first instance there results denervation of the smooth muscles of the eye supplied by the sympathetic nervous system and hypersensitiveness to epinephrine. In the second case no denervation and no hypersensitiveness follow.

The close relation of nasopharyngeal tumors to the base of the skull and their tendency to invade cause lesions of the cranial nerves, especially those of the eye. In the presence of a Horner's syndrome which cannot be accounted for by a

neurological examination, the epinephrine test may be the only deciding factor in the diagnosis. At any rate, it will give additional information when the objective examination is not conclusive.

NEUROLOGY OF THE NOSE.

Headache: For purely clinical purposes Proetz[15] advises the following classification of headaches:

Having *A.* definite demonstrable causes;

B. semidemonstrable causes;

C. undemonstrable or only remotely suggestive causes.

Obviously Class C is the most interesting group because it comprises not only the most frequent but also the most perplexing cases. It is important to remember that less than 5 per cent of headaches are referable to paranasal sinuses no matter what their location, and that the great majority are due to local vascular disturbances, whatever their remote cause might be.

When pressure changes occur in the vascular structure of the brain, the pain is likely to occur during the relaxation and dilatation which follow pressure, and not during the elevation of pressure; therefore, the treatment must primarily consist of experimental alteration of the vascular tone. Unless there is hypertension, the patient is given ephedrine orally (together with seconal or some other barbiturate to minimize the unpleasant effects). The ephedrine is given in doses of three-eighth grain twice daily and if effective is continued for a week. If, after the drug is withdrawn, the headache returns, the use of ephedrine may be resumed, but a long continued course of this drug is not recommended. Some persistent headaches can be controlled with occasional courses of ephedrine.

If this is unsuccessful, and especially if the patient complains of lassitude and fatigue, thyroid extract takes the place of ephedrine. Thyroid may defeat the headache even when the basal metabolic rate is normal or slightly increased. If this succeeds, then the dosage is subsequently determined by the symptomatology rather than the metabolic rate. Thy-

roid may be administered at the same time with thiamine. Of great importance is evacuation of the lower bowel, preferably with an enema, at the very onset of an attack of headache, regardless of the patient's statements that his habits are regular.

The original paper is recommended for further details.

Injury of the Paranasal Sinuses: Goldman[7] reports the case of a male patient, 40 years of age, who received a bomb injury 22 months prior to admission. Ophthalmological examination showed total blindness and absence of light perception in the right eye. The vision in the left eye was reduced to counting of fingers at one foot, corrected with +1.50 sphere to 20/70. The right eye could not be moved beyond the midline on looking to the right, nor could it be depressed. The right pupil was in mid-dilatation, irregular, did not react to light, but reacted slightly consensually and during convergence. The left pupil was normal in size, reacted to light, but reacted only slightly consensually. Fundus examination showed the right disc to be pale and grayish. Below and temporal to the discs there was a linear area of chorioretinitis. The field of vision of the left eye was constricted to within 20° of the point of fixation and was of the tubular type. The color of a 3 mm. object could not be recognized but all of the 10 mm. color test objects were easily discernible. The corrected vision in the left eye was 20/70. An intranasal operation was performed and a shrapnel was removed from the left sphenoid and left posterior ethmoid. Immediately following removal of the foreign body, and while the patient was still on the operating table, he exclaimed that he could see. Finger-counting tests confirmed this. The prompt restoration of vision suggested the possibility that one was dealing with hysterical amblyopia. On the other hand, removal of pressure of the sharpnel on the lateral wall, especially where there has been loss of wall substance due to pressure atrophy, particularly in the presence of a projecting optic nerve, would explain the rapid return of vision. At any rate, the patient was definitely cured and was able to resume his occupation.

NEUROLOGY OF THE EAR.

Internal Ear: Altmann and Fowler[1] present a detailed description of the histological findings of the internal ear in

three instances of Ménière's syndrome. The findings were strikingly similar to those in previously published cases. In all of these cases a dilatation of the endolymphatic system was the most prominent feature. The extent of the dilatation varied. Dilatation of the cochlear duct was always present; the semicircular canals remained free from dilatation. The end-organs in the three cases showed postmortal but no other definitely pathological changes. Disturbances of the secretion of the endolymph are probably the primary cause with fibrosis of the "perisaccular" tissue in the area of the endolymphatic sac as a predisposing factor. Among the surgical procedures, opening of the endolymphatic sac seems at present to be the most promising approach.

Schick[16] advises intravenous injections of 5 cc. of a 50 per cent magnesium sulfate solution as treatment for Ménière's syndrome. The injections are given two or three times weekly, and 10 to 20 injections usually suffice. In some cases treatment was repeated after a few months. The injections should be performed at an extremely slow rate with the patient in the recumbent position, in order to decrease the annoying sensation of heat in the head. Among 18 patients, the following results were obtained: In seven cases no spells occurred during a control period of 14 months. In seven cases marked improvement occurred; *viz.*, the spells were less frequent and milder, there was no more dizziness in the intervals, the tinnitus decreased and the hearing was better. In four cases there was no change.

Forbes[5] reports the following statistics: In a series of 1,401 cases of all types of deafness, 316 were cases of nerve deafness and 1,085 other types of deafness. In 109 of the 316 cases of nerve deafness there was a history of the ingestion of quinine over a considerable period of time; in 22 of the 109 cases the patients regarded quinine as the cause of the deafness; in seven they ascribed their sudden and permanent impairment of hearing to a single course of therapy with large doses of quinine. In only 93 of the 1,085 cases of all other types of deafness was there a history of the ingestion of quinine, and in only two cases was the deafness even associated with this drug by the patients. Thus, in 34 (5 per cent) of the cases of nerve deafness, quinine was a significant factor while in only 8.5 per cent of the much larger group of cases of all other types of the deafness was it considered.

The intake of large doses of quinine is not required to cause deafness, as idiosyncrasy for this drug is an important consideration in the etiology of nerve deafness. Quinidine, another frequently used alkaloid of cinchona, may also be a cause of nerve deafness.

Brunner[2] presents a review of the physiology of the labyrinth and its usefulness in examining aviators. He states that the semicircular canals of the human labyrinth by no means respond accurately to rotating motions of the body, their threshold being rather high; therefore, the semicircular canals are far inferior to the eye in this respect, and they are not of great help to the pilot in performing a spin. There is even a possibility that the functon of the normal semicircular canals, by causing vertigo, might become dangerous for the pilot after having performed a spin.

A normal individual also does not perceive accurate information from the otoliths concerning his position in the space. This holds true even when the otoliths co-operate with the "deep sensibility" of the body. The most accurate spatial knowledge is obtained by direct vision.

Summarizing, one may say that the function of the normal labyrinth is of little help to the aviator. The pilot's pilot is the eye, unless its function is disturbed by the influence of the labyrinth upon the external eye muscles, because only the eye measures exactly the degree of tilt of the body or the rate and direction of body rotation. The labyrinth may offer actually false information — illusions which occasionally might lead to disaster. These illusions are very disturbing to the pilot, as his instruments give him definite information, but his labyrinth may give him contradictory information when flying blind. The successful blind flyer will correlate his senses with his instruments. He may learn this correction by proper education and correct judgment, the latter depending on his normal mental activity.

The conditions are quite different if the pilot experiences not only illusions but actual vertigo. Vertigo is not simply an illusion of a position or a motion which can be recognized as such by a trained aviator and which does not mean a loss of orientation, but simply a false orientation. Vertigo rather appears if the abnormal activity of the brain, leading to the

illusion, spreads over the vasovegetative centers and finally spreads over the higher centers of mental activity, disturbing the consciousness, the ability of correct judgment, et cetera, and occasionally, but not frequently, terminating in a blacking out. If a pilot experiences illusions and simultaneously his ability for proper judgment is diminished or even lost, he necessarily faces disaster. This fact is of great importance as there is no habituation to vertigo due to rotation of body, as far as practical conditions are concerned.

Unfortunately, the practical results obtained by the usual labyrinthine tests, particularly by the turning chair test, were extremely unsatisfactory, up to the present time; however, a renewed consideration of the entire problem should be attempted, taking advantage of the modern methods of functional test. In examining the labyrinths of a flying cadet, one is not actually interested in the function of the labyrinths *per se;* much more important is the susceptibility to vertigo. By susceptibility to vertigo is meant that the candidate will respond with vertigo upon stimuli of minor intensity which do not produce vertigo in average individuals; however, the diagnosis of labyrinthine hyperexcitability is difficult as there is no exact method for that diagnosis at our disposal. As far as this diagnosis is concerned, two facts have to be kept in mind: First, the duration of the post-turning nystagmus is not a certain indicator of the post-turning vertigo; second, the turning test is such a powerful test that the diagnosis of hyperexcitability should not be made by that test. The turning test, therefore, according to the technique of Bárany, must be considered as the least suitable of all labyrinthine tests for the diagnosis of labyrinthine hyperexcitability, and we should better apply stimuli of minor intensity in order to examine the sensitivity of the labyrinths. It is not necessary that these stimuli be adequate to the physiology of the labyrinths, namely, that they consist of motions of the head or body; the stimuli might be also inadequate, inasmuch as the labyrinths of aviators frequently are stimulated by inadequate stimuli as changes of the intratympanal pressure or vasomotor disturbances or aeroembolism. The author feels that the caloric test, according to the technique of Kobrak, has definite advantages in this respect: It permits a more exact diagnosis of labyrinthine hyperexcitability, it is not dis-

agreeable for the candidate, it takes no more time than the turning test, and it does not require special instruments.

Facial Paralysis: McCall and Gardiner[12] report three cases of facial paralysis following mastoid surgery. In the first case a facial paralysis appeared eight days after a radical mastoid operation. The paralysis disappeared after galvanic stimulations of the muscles, heat and gentle massage over a period of one month.

In the second case the facial paralysis appeared immediately after a radical mastoid operation. Four days after the operation the facial nerve was uncovered for 1 cm. from its course in the middle ear to its disappearance deep in the facial ridge. Two depressed bone chips in the facial canal were found causing pressure on the nerve, opposite the horizontal semicircular canal. The bone chips were removed, galvanic stimulation, heat and massage were applied to the paralyzed facial muscles, and approximately five months after decompression, slight movements returned to the right side of the face.

In the third case a facial paralysis followed a simple mastoid operation. The facial canal was exposed and the facial nerve was found to be severed about 2 mm. below the horizontal semicircular canal, with loss of three-quarters of a centimeter of the nerve. A neuroma on the proximal end of the severed nerve was removed and this as well as the distal end was freshened up. Next, a nerve graft obtained by exposing the lateral femoral cutaneous nerve was placed in the bony canal, approximating the freshened proximal and distal ends of the open facial nerve. Nine months later the boy was able to move the previously paralyzed corner of his mouth. Gradually the paralyzed muscles, with exception of the frontalis, have assumed a functioning activity. Unfortunately, the authors do not present pictures showing the patient prior to the operation.

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ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES.

Representatives from 14 foreign countries will attend the fifty-first annual convention of the Association of Military Surgeons of the United States, the great symposium on war medicine to be held in Philadelphia, Oct. 23.

Lieut.-Gen. Sir Alexander Hood, director-general of the British Army Medical Services, will head the list of visiting medical men from abroad. Lieut.-Gen. Hood will make the principal address at the opening night's session of the military surgeons' meeting in the ballroom of the Bellevue-Stratford Hotel.

The three-day convention, the most important medical meeting ever held in this country in wartime, will be attended by 2,000 doctors now attached to the armed forces here and abroad, as well as by the thousands of other physicians and medical men in civilian practice.

With Lieut.-Gen. Hood in the British delegation will be Maj.-Gen. C. Max Page and Col. Frank S. Gillespie, of the Royal Army Medical Corps, at present liaison officer at the Medical Field Service School, Carlisle Barracks, Pa.

Canada will be represented by Brig. G. B. Chisholm, director-general of medical services of the Royal Canadian Army; Surgeon Capt. A. McCallum, director general of medical services for the Royal Canadian Navy; Wing Comdr. H. A. Peacock, principal medical officer of the First Training Command; Wing Comdr. L. M. Emard, principal medical officer of the Third Training Command; and Lieut.-Col. T. A. Lebbetter, of the Royal Canadian Army.

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